U.S. Department of Labor

Office of Administrative Law Judges
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Issue date: 30Mar2001

Case No: 2000-BLA-0637

In the Matter of

JACK R. BLANKENSHIP,
Claimant

v.

KENTUCKY CARBON CORPORATION,
Employer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party-in-Interest

APPEARANCES:

William Lawrence Roberts, Esq.
For the claimant

Natalie Brown, Esq.
For the employer

BEFORE: JOSEPH E. KANE
Administrative Law Judge

DECISION AND ORDER - AWARDING BENEFITS

This proceeding arises from a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, 30 U.S.C. § 901 *et seq.* (the Act). Benefits are awarded to coal miners who are totally disabled due to pneumoconiosis. Surviv-ing dependents of coal miners whose deaths were caused by pneumoconiosis may also recover benefits. Pneumoconiosis, commonly known as black lung, is a chronic dust disease of the lungs arising from coal mine employment. 20 C.F.R. § 718.201 (1996).

On April 17, 2000, this case was referred to the Office of Administrative Law Judges for a formal hearing. Following proper notice to all parties, a hearing was held on November 15, 2000 in Pikeville, Kentucky. The Director's exhibits were admitted into evidence pursuant to 20 C.F.R. §725.456, and the parties had full opportunity to submit additional evidence and to present closing arguments or post-hearing briefs. The Employer's Closing Argument was received on December 14, 2000.

The Findings of Fact and Conclusions of Law that follow are based upon my analysis of the entire record, arguments of the parties, and the applicable regulations, statutes, and case law. They also are based upon my observation of the demeanor of the witnesses who testified at the hearing. Although perhaps not specifically mentioned in this decision, each exhibit and argument of the parties has been carefully reviewed and thoughtfully considered. While the contents of certain medical evidence may appear inconsistent with the conclusions reached herein, the appraisal of such evidence has been conducted in conformance with the quality standards of the regulations.

The Act's implementing regulations are located in Title 20 of the Code of Federal Regulations, and section numbers cited in this decision exclusively pertain to that title. References to DX, CX and EX refer to the exhibits of the Director, the claimant, and the employer, respectively. The transcript of the hearing is cited as "Tr." and by page number.

ISSUES

The following issues remain for resolution:

1. whether the claimant has pneumoconiosis as defined by the Act and regulations;

2. whether the claimant's pneumoconiosis, if any, arose out of coal mine employment;

3. whether the claimant is totally disabled;

4. whether the claimant's disability is due to pneumoconiosis;

5. whether the evidence establishes a change in conditions or a mistake in a determination of fact within the meaning of 20 C.F.R. § 725.310; and

6. whether the evidence establishes a material change in conditions within the meaning of 20 C.F.R. § 725.309(d).

(Tr. 8-9; DX 74).

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Factual Background and Procedural History

The claimant, Jack R. Blankenship, was fifty-nine years old at the time of the hearing and has a seventh grade education. He has one dependent, his wife, for purposes of augmentation of benefits. (Tr. 10, 15, 21-22; DX 1, 5).

The claimant began working as a coal miner at the age of sixteen. All of his coal mine employment was underground at the face. The claimant ceased working on October 15, 1982 due to health problems. His last position was section foreman. (Tr. 10-11, 16; DX 1). The parties previously stipulated that the claimant had been a coal miner for at least ten years. (DX 56, 61, 74).

The claimant filed his first claim for black lung benefits on December 16, 1983. (DX 23.413). Following a formal hearing on May 17, 1989 (DX 23.67), the claim was denied by Administrative Law Judge Daniel J. Roketenetz on July 23, 1990. Judge Roketenetz found, utilizing the "true doubt" rule, that the claimant established the existence of simple pneumoconiosis and that his pneumoconiosis arose out of his coal mine employment. However, he found that the claimant had not established total disability, and therefore benefits were denied. (DX 23.53). By decisions entered September 17, 1991, and September 9, 1992, the Benefits Review Board ("the Board") and the United States Court of Appeals for the Sixth Circuit affirmed Judge Roketenetz's Decision and Order. (DX 23.15, 23.4).

The claimant filed the instant duplicate claim on January 26, 1995. (DX 1). The employer was notified of the claim, and subsequently controverted based on both its liability and the claimant's eligibility. (DX 19, 20, 21, 22). The claim was denied by the District Director, Office of Workers' Compensation Programs ("OWCP") on July 13, 1995. (DX 15). The claimant timely requested a formal hearing, and the claim was forwarded

to the Office of Administrative Law Judges ("OALJ") on October 18, 1995. (DX 16, 24). Following a formal hearing on December 3, 1996, Administrative Law Judge J. Michael O'Neill

denied benefits on December 9, 1997. (DX 53, 56). Judge O'Neill found that the objective test results and the Claimant's testimony given at the hearing before Judge Roketenetz on May 17, 1989 (DX 23.94, 23.96) contradicted the claimant's more recent testimony that he smoked only a few cigarettes in his lifetime. Judge O'Neill determined that the preponderance of the x-ray evidence was negative for pneumoconiosis and that the medical opinion evidence failed to establish the existence of pneumoconiosis. Although he found that the evidence established total disability from a respiratory standpoint, he did not find that the impairment was due to pneumoconiosis. (DX 56). On appeal, the Board affirmed Judge O'Neill's Decision and Order on December 18, 1998. (DX 61).

On November 19, 1999, the claimant filed a request for modification, supporting his request with the November 4, 1999 report of Dr. R. Sundaram. (DX 62). The OWCP issued a proposed Decision and Order denying the request for modification on January 11, 2000. (DX 66). The claimant again timely requested a formal hearing, and the claim was forwarded to the OALJ on April 17, 2000. (DX 64, 67, 69, 74).

Medical Evidence

The following is a summary of the medical evidence submit-ted with the instant duplicate claim and subsequent request for modification:

A. Chest X-rays

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications[1]	Interpretation
DX 43	5/2/94	3	Scott/BCR, B	0/1, p/p, upper and mid zones. Non-specific scattered linear fibrosis. Minimal bullous emphysema apices. Hyperinflation lungs compatible with emphysema. Enlargement upper mediastinum?
DX 46	5/2/94	3	Wiot/BCR, B	No CWP. Most likely sarcoidosis; could be IPF.
DX 43	5/2/94	3	Wheeler/BCR, B	No CWP or silicosis. Probable subtle non-specific linear interstitial fibrosis in portion both lungs. Probable emphysema with areas of decreased lung markings.

[1] The symbol "BCR" denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc. or the American Osteopathic Association. 20 C.F.R. § 727.206(b)(2).

The symbol "B" denotes a physician who was an approved "B-reader" at the time of the x-ray reading. A B-reader is a physician who has demonstrated expertise in assessing and classifying x-ray evidence of pneumoconiosis. These physicians have been approved as proficient readers by the National Institute of Occupational Safety & Health, U.S. Public Health Service pursuant to 42 C.F.R. § 37.51 (1982).

DX 32	5/6/94	-	Amim	Severe chronic obstructive lung disease. Diffuse fine interstitial fibrosis.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 46	8/25/94	2	Wiot/BCR, B	No CWP. Sarcoidosis or IPF.
DX 43	8/25/94	2	Wheeler/BCR, B	No CWP or silicosis. Probable subtle non-specific linear interstitial fibrosis. Probable emphysema with areas of decreased lung markings.
DX 43	8/25/94	2	Scott/BCR, B	0/1, p/p, upper and mid zones. Scattered non-specific fibrosis. Emphysema with hyperinflation lungs. Minimal bullous emphysema apices. Enlargement upper mediastinum?
DX 11	2/27/95	-	Mettu	2/2, q/q.
DX 13	2/27/95	3	Sargent/BCR, B	1/0, s/p, six zones; could be due to disease process other than pneumoconiosis. Peural thickening. Smoking history?
DX 14	2/27/95	1	Poulos/BCR, B	2/2, q/q, six zones. Pleural thickening.
DX 25	2/27/95	U/R	Wiot/BCR, B	Unreadable.
DX 30	2/27/95	2	Wiot/BCR, B	No CWP. Sarcoidosis.

DX 25	2/27/95	U/R	Spitz/BCR, B	Unreadable.
DX 32	2/27/95	2	Spitz/BCR, B	No CWP; possible idiopathic pulmonary fibrosis or sarcoidosis.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 27	9/8/95	1	Sargent/BCR, B	½, s/s, six zones; etiology unknown, not CWP. Smoking history? Subpleural fat.
DX 30	9/8/95	2	Wiot/BCR, B	No CWP. Sarcoidosis.
DX 22A	9/8/95	2	Rubenstein/ BCR, B	3/2, s/s, six zones. Hi.
DX 44	9/8/95	1	Mathur/BCR, B	2/3, p/s, six zones.
DX 32	9/8/95	2	Spitz/BCR, B	No CWP. Possible IPF or sarcoidosis.
DX 43	9/12/95	1	Scott/BCR, B	0/1, p/p, upper and mid zones. Non-specific linear interstitial fibrosis. Emphysema with minimal bullous emphysema apices. Lateral pleural thickening; could this be due to steroid treatment? Same for mediastinum.
DX 43	9/12/95	1	Wheeler/BCR, B	No CWP or silicosis. Minimal nonspecific linear interstitial fibrosis. Emphysema with areas of decreased lung markings.

DX 22B	9/12/95	-	Dahhan/B	½, p/p, upper and mid zones. Wide mediastinum.
DX 26	9/12/95	1	Wiot/BCR, B	No CWP. Sarcoidosis or IPF.
DX 26	9/12/95	1	Spitz/BCR, B	No CWP. May be IPF or sarcoidosis. Subpleural fat deposition.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 26	9/12/95	1	Shipley/BCR, B	2/1, s/t, six zones; not CWP. Diffuse interstitial lung disease with probable hilar and mediastinal lymph adenopathy.
DX 34	4/23/96	2	Hippensteel/B	2/2, p/s, mid and lower zones. Pleural thickening.
DX 46	4/23/96	2	Wiot/BCR, B	No CWP. Sarcoidosis or IPF.
DX 43	4/23/96	1	Wheeler/BCR, B	No CWP or silicosis. Minimal nonspecific linear interstitial fibrosis. Emphysema with areas of decreased lung markings in upper lobes. Discoid atelectasis above left CPA.
DX 43	4/23/96	1	Scott/BCR, B	0/1, p/p, upper and mid zones. Non-specific interstitial linear fibrosis. Emphysema with apical bullous changes. Lateral pleural

				thickening and possible mediastinal enlargement enlargement which could be fat due to steroid therapy. Minimal discoid atelectasis near left CPA.
DX 40	5/29/96	-	Halbert/BCR, B	Increased interstitial markings bilaterally, presumed to be chronic. Mild prominence of upper mediastinum which appears slightly widened.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 45	5/29/96	1	Fino/B	No CWP. Diffuse increase in lung markings.
DX 45	5/29/96	3	Wheeler/BCR, B	No silicosis or CWP. Minimal right paratracheal adenopathy. Emphysema with areas of decreased lung markings in upper lobes.
DX 42	5/29/96	3	Wiot/BCR, B	No CWP. Sarcoidosis.
DX 47	5/29/96	2	Castle/B	½, t/s, mid and lower zones; consistent with asbestosis, not CWP. Could be sarcoidosis. Pleural thickening.
DX 48	5/29/96	1	Dahhan/B	2/2, s/t, six zones.
EX 16	5/29/96	1	Hippensteel/B	2/2, p/t, mid and lower zones. Pleural thickening. Right hilar adenopathy. Findings are

				compatible with sarcoidosis. Not typical for CWP.
DX 41	6/4/96	-	Halbert/BCR, B	Increased interstitial markings. Mild prominence of upper mediastinum.
DX 62	11/4/99	1	Sundaram	½, p/q, upper and mid zones.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 70	11/4/99	3	Wiot/BCR, B	No CWP. Lung fields are markedly overexpanded, consistent with emphysema. Changes RUL consistent with bronchiectasis and past inflammatory disease. Fine granular pattern to the mid lung fields bilaterally, not due to coal dust, but etiology unknown. Questionable pleural plaque along right lateral chest wall.
DX 72	11/4/99	3	Shipley/BCR, B	No CWP. Diffuse interstitial lung disease, probably interstitial pulmonary fibrosis. Right hilar and medias-

				tinal changes are suspicious for malignancy.
DX 73	11/4/99	2	Spitz/BCR, B	No CWP. Linear strands RUL and LML laterally may be due to a previous inflammatory process. Bilateral pleural plaques may be due to previous asbestos exposure. The cause of the hazy appearance in the mid portion of the lungs is not unknown.
EX 16	11/4/99	3	Hippensteel/B	½, p/t, mid and lower zones. Pleural thickening. Right hilar adenopathy. Findings compatible with sarcoidosis. Not typical for CWP.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
CX 2	12/10/99	-	West	Stable chest without active changes.
DX 65	12/18/99	1	Dahhan/B	0/0. Co. Emphysema.
DX 70	12/18/99	2	Spitz/BCR, B	No CWP. Pleural plaques which may be secondary to previous asbestos exposure.
EX 1	12/18/99	3	Scott/BCR, B	Minimal diffuse non-specific linear interstitial fibrosis. Minimal bullous emphysema apices. Probable sub-

pleural fat lateral chest walls.

EX 1	12/18/99	3	Wheeler/BCR, B	SUL increased lung markings. Probable tiny linear scar LUL. Possible subtle bilateral apical pleural thickening. Minimal smooth pleural fibrosis or small pleural plaque if there has been asbestos exposure.
DX 72	12/18/99	3	Shipley/BCR, B	No CWP. Diffuse irregular opacities consistent with interstitial lung disease, possibly interstitial pulmonary fibrosis. Possible left pleural plaque vs. extra-pleural fat deposition.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
DX 72	12/18/99	3	Wiot/BCR, B	No CWP. Extensive emphysema. Pleural thickening along left lateral chest wall, localized, consistent with a pleural plaque.
EX 3	12/18/99	3	Wheeler/BCR, B	Subtle increased lung markings. Probable tiny linear scar in lower lateral periphery LUL. Possible subtle bilateral

				apical pleural thickening. Minimal smooth pleural fibrosis or small pleural plaque on left lateral chest wall if there has been asbestos exposure.
EX 16	12/18/99	2	Hippensteel/B	2/1, p/t, mid and lower zones. Pleural thickening. Right hilar adenopathy. Findings compatible with sarcoidosis. Not typical for CWP.
EX 3	7/10/00	2	Wheeler/BCR, B	Subtle linear increased mid and lower lung markings compatible with pulmonary vascular prominence accentuated by underexposure or early interstitial fibrosis or infiltrates. Possible few small nodules in left lateral CPA. Probable minimal decreased upper lung markings compatible with emphysema.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
EX 3	7/10/00	2	Scott/BCR, B	Emphysema with bullous changes upper lungs. Non-specific linear interstitial fibrosis mid and lower lungs. Subpleural fat lateral chest walls. Few nodules LLL: granulomata versus

				metastases.
EX 14	7/10/00	1	Wiot/BCR, B	Lung fields are somewhat overexpanded, consistent with emphysema. Pleural changes, which may represent pleural plaques, although this may only represent deposition of subpleural fat. Within RUL, there is some stranding extending to the right hilum, consistent with bronchiectasis. Fine granular pattern within mid and lower zones, unrelated to coal dust exposure. Etiology of this process cannot be determined.
EX 7	7/10/00	1	Broudy/B	2/3, s/p, mid and lower zones. Pleural thickening. Several scattered calcified granulomas. More likely asbestosis than pneumoconiosis.

Ex.No.	Date of X-ray	Film Qual.	Physician/ Qualifications	Interpretation
EX 5	7/10/00	1	Castle/B	2/1, t/s, mid and lower zones. Pleural thickening. Possible hilar adenopathy. Changes

				present are not consistent with CWP. More likely are due to asbestosis or non-occupational disease such as sarcoidosis.
EX 2	7/10/00	1	Hippensteel/B	2/1, p/t, mid and upper zones. Pleural thickening. No distinct hilar adenopathy visible but findings could be from sarcoidosis.

B. Pulmonary Function Studies

Date	Ex. No.	Age/Hgt.	FEV1	FVC	FEV1/ FVC	MVV	Coop/ Comp.
5/9/94	DX 8[2]	53/70"	67%	68%	- -	- -	- -
6/16/95	DX 6	54/70"	2.00 * 3.60	3.41 2.50	- -	- -	Fair/ Good

Dr. Steve Kraman reviewed the June 16, 1995 study and found it to be invalid. He stated that Dr. Fritzhand incorrectly calculated the FEV1 value which should have been 2.00. He also stated that the claimant's effort was uneven and questioned the adequacy of the claimant's effort. (DX 6).

Date	Ex. No.	Age/Hgt.	FEV1	FVC	FEV1/ FVC	MVV	Coop/ Comp.

[2] Dr. Younes did not provide the recorded values. Rather, he gave only percentages of the predicted values. Moreover, only one trial was conducted.

7/7/95	DX 7	54/70"		2.18	2.98	73%	- -	Good/
			*	2.51	3.11	81%		Good

Dr. Younes, who obtained the July 7, 1995 study, stated that the claimant could not exhale longer than three to four seconds and coughed. (DX 7). He concluded that the study was invalid.

Dr. N.K. Burki reviewed the study and determined that it was invalid due to less than optimal effort, cooperation and comprehension. (DX 7).

9/8/95	DX 22A	54/69"		2.30	3.40	- -	- -	Good/
			*	2.50	3.60			Good

Dr. Fritzhand noted that the claimant was unable to complete the MVV due to coughing.

9/12/95	DX 22B	54/68.75"		2.04	3.25	63%	65.28	Fair/
			*	2.24	3.55	63%	41.66	Good

Dr. Dahhan, who obtained the September 12, 1995 study, stated that the claimant did not give consistent effort and did not continue expiration for six seconds nor reach a plateau. He also stated that the claimant's effort on the MVV was not maximal.

4/23/96	DX 34	55/67"		2.04	2.74	75%	91.0	Good/
			*	2.14	2.91	74%	84.0	Good
7/24/96	DX 40, 41	55/70"		- -	- -	- -	- -	
			*	1.89[3]	3.39			
12/18/99	DX 65	58/68"		2.18	3.57	61%	56.6	Poor/
			*	1.73	2.77	62%	44.67	Good

[3] Judge O'Neill calculated the FEV1 value of this study by the listed FVC value of 3.39 and the listed FEV1/FVC ratio of 50.60. It appears that the results were collected after bronchodilation, although Dr. Saloum's report states that both pre- and post-bronchodilation studies were obtained. Only one set of results appears in the report.

Date	Ex. No.	Age/Hgt.		FEV1	FVC	FEV1/ FVC	MVV	Coop/ Comp.
12/22/99	CX 1	58/70"		1.95	2.67	72.84%	- -	- -
			*	1.43	1.98	72.43%		

Dr. De Joya commented that the December 22, 1999 study showed a moderate to severe restrictive lung defect without significant bronchodilator response, and moderate to severe small airways disease. No tracings were provided.

7/10/00	EX 2	59/69"		1.75	2.79	63%	44	Poor/
			*	1.90	3.06	62%		Good

Dr. Hippensteel stated that the July 10, 2000 study was invalid due to suboptimal effort.

* Results obtained post-bronchodilator.

C. Arterial Blood Gas Tests

Date	Physician		pCO2	pO2	Ex. No.
5/9/94	Younes		31.5	73.8	DX 8
2/27/95	Mettu		34.8	72.8	DX 12
9/8/95	Fritzhand		33.4	84.3	DX 22A
9/12/95	Dahhan		33.3	68.8	DX 22B
		**	32.6	62.7	
4/23/96	Hippensteel		34.4	61.8	DX 34
12/18/99	Dahhan		34.1	69.5	DX 65
		**	32.0	82.0	
7/10/00	Hippensteel		34.9	70.6	EX 2

** Results obtained with exercise.

D. CT scans and Medical Opinions

Dr. Maan Younes was the claimant's treating physician. Progress notes from Dr. Younes, spanning May 2, 1994 to March 30, 1995,

are in the record. Those notes reveal diagnoses of s/p chronic obstructive pulmonary disease with exacerbation which improves with steroids, bronchodilators and antibiotics; coal workers' pneumoconiosis; bilateral chest pain, most likely secondary to chronic coughing; history of coronary artery disease; PUD; arthritis; severe chronic bronchitis; bilateral hilar and mediastinal hyphadenopathy, r/o coal workers' pneumoconiosis; dizziness and light headedness, r/o intracranial lesion; r/o lymphoma; r/o infectious process (TB); paraseptal emphysema; headaches and visual changes; history of peptic ulcer disease;
OA right shoulder; and persistent HAS. Dr. Younes noted that the claimant quit smoking in approximately 1979, having been a light smoker who smoked for a short period of time. (DX 8). Dr. Younes referred the claimant for a CT scan on May 6, 1994. It was read by Dr. Ansuya Amim as showing severe chronic lung disease with enlarged lymphadenopathy in paratracheal region, carina and right hilar region. Diffuse fine interstitial fibrosis was noted bilaterally, as was pleural thickening. Dr. Amim suggested a bronchoscopy for better evaluation. (DX 32). A bronchoscopy in 1994 was negative for malignancy and infection. (DX 8).

Dr. Younes also issued a series of letters regarding the claimant's medical condition. On May 16, 1995, Dr. Younes stated that the claimant "has unequivocal evidence of Coal Workers' Pneumoconiosis as evidenced by the finding on his chest x-ray." He stated that based on the claimant's twenty-three years of coal dust exposure, physical examination and x-ray, the claimant has pneumoconiosis. He also stated that the claimant has severe chronic obstructive pulmonary disease. In a letter dated April 15, 1996, Dr. Younes stated that the claimant has severe chronic obstructive pulmonary disease and severe coal workers' pneumoconiosis with persistent wheezing and dyspnea on minimal exertion. He also noted insulin dependent diabetes. Dr. Younes stated that "I have no question in my mind that he does have severe Coal Workers' Pneumoconiosis as evidenced by the findings on his chest x-ray." He attributed a substantial portion of the claimant's chronic obstructive pulmonary disease to coal dust exposure. In a letter dated May 14, 1996, Dr. Younes stated that the claimant is permanently and totally disabled from a pulmonary standpoint as a result of chronic obstructive pulmonary disease and coal workers' pneumoconiosis, and that he felt that a great part of the respiratory disability arose out of the claimant's coal mine employment. Dr. Younes also completed an insurance company form stating that the claimant is permanently disabled, referring to both his lung condition and diabetes. (DX 10, 35, 38, 50).

Dr. R.V. Mettu examined the claimant on February 27, 1995. He recorded twenty-six years of coal mine employment, a family history of high blood pressure, heart disease, diabetes, and

cancer, and a medical history of pneumonia, wheezing, arthritis, heart disease, and three heart attacks. He noted a smoking history of one-fourth pack of cigarettes per day from 1951 to 1965. The claimant complained of a cough with sputum, wheezing, dys-pnea, and chest pain. Dr. Mettu interpreted an x-ray as 2/2, q/q. He diagnosed chronic bronchitis due to symptoms consistent with chronic bronchitis, including scattered rhonchi. He also diagnosed pneumoconiosis by x-ray, arthritis, and a history of arteriosclerotic heart disease. Dr. Mettu did not make an assessment of the claimant's pulmonary function because administration of a study was pending. (DX 11). Although a supplemental report was supposed to be issued, no such report appears in the record.

Dr. Martin Fritzhand interviewed and examined the claimant on September 8, 1995. He noted symptoms of shortness of breath, a cough with sputum, and chest pain. He also noted nine years of coal mine employment with Kentucky Carbon Corporation, working as a section foreman, and that the claimant was a non-smoker. He also noted that the claimant stated that he had obtained multiple x-rays over the years that were positive for pneumoconiosis. Dr. Fritzhand diagnosed pneumoconiosis and r/o occult pulmonary disease. He stated that there is an adequate history of coal dust exposure and x-ray changes consistent for a diagnosis of pneumoconiosis, and that coal dust alone could explain many of the claimant's symptoms. Based on the pulmonary function study results, Dr. Fritzhand noted mild to moderate chronic obstructive airways impairment. He stated that the claimant certainly has an impairment of respiratory function and would be unable to return to his last coal mine employment. He stated that the claimant appeared totally and permanently disabled. He noted that this was especially prominent because the claimant was a non-smoker. (DX 22A).

Dr. Abdul K. Dahhan examined the claimant on September 12, 1995 on behalf of the employer. He recorded the claimant's symptoms of a cough with sputum, wheezing, dyspnea on exertion, and chest pain; his medical history of back pain, diabetes mellitus, and heart attacks; and his occupational history of twenty-three years of coal mine employment. Dr. Dahhan noted that the claimant smoked a short time as a teenager. An x-ray was read as ½, p/p. He administered a pulmonary function study during which the claimant gave less than optimal effort. The arterial blood gas test revealed mild hypoxia. The carboxyhemoglobin level was 10.3%, consistent with a person

smoking two packs of cigarettes per day. Dr. Dahhan found sufficient radiological findings to justify a diagnosis of coal workers' pneumoconiosis. He also diagnosed obstructive airways disease. Dr. Dahhan concluded that the claimant does not retain the physiological capacity to perform heavy manual labor, based on Dr. Younes's statement that the claimant continues to have episodes of bronchospasms requiring continuous treatment with steroids. He stated that the cause of the claimant's pulmonary impairment is hyperactive airway disease manifested by bronchial asthma. He determined that it was not due to coal dust. He noted that the coal workers' pneumoconiosis was not sufficient to cause the development of a respiratory impairment or disability. Dr. Dahhan also reviewed other medical records when issuing this opinion, including his previous examination report. (DX 22B). Dr. Dahhan is board-certified in internal and pulmonary medicine. (DX 33; EX 13).

Dr. Bruce Broudy issued a consultative report on November 20, 1995, on behalf of the employer. He reviewed medical records, including his examination report of September 12, 1984. Dr. Broudy determined that there was sufficient objective evidence to justify a diagnosis of coal workers' pneumoconiosis. He also diagnosed chronic obstructive airways disease due to cigarette smoking. Dr. Broudy noted that although the claimant contended that he had smoked very little, there was objective evidence of continual smoking as late as September 1995 at Dr. Dahhan's examination. He attributed the restrictive defect on pulmonary function study testing to poor effort. He found that the studies of March 21, 1989, April 3, 1989, June 16, 1995, and July 7, 1995, were invalid. He reviewed medical reports from Drs. Clarke, Baxter, Guberman, Anderson, Myers, Penman, O'Neill, Dahhan, Wright, Fritzhand, Mettu, Younes, and Quillan. Dr. Broudy concluded that the claimant was not totally disabled. He explained that the results of valid pulmonary function studies showed only a mild impairment and exceeded federal disability guidelines. Dr. Broudy attributed any respiratory impairment present to smoking. (DX 26). Dr. Broudy is board-certified in internal and pulmonary medicine. (DX 33; EX 13).

Dr. Gregory J. Fino reviewed medical records and issued a report on December 7, 1995 on behalf of the employer. Dr. Fino mentioned that in two previous reports he found the claimant not disabled due to pneumoconiosis, but possibly disabled due to coronary artery disease. Dr. Fino stated that if the claimant had no respiratory impairment due to coal dust when he left the mines, he would not develop such an impairment later. He also noted that the claimant developed a significant impairment in oxygen transfer and an obstructive ventilatory abnormality during a period in which he was

not exposed to coal dust. Dr. Fino thus concluded that pneumoconiosis had not caused the claimant's pulmonary impairment. He concluded that the claimant's pulmo-nary impairment is sufficient enough to cause a pulmonary disability and is related entirely to smoking. He further noted that the claimant's simple coal workers' pneumoconiosis had not progressed. (DX 28). Dr. Fino is board-certified in internal and pulmonary medicine. (DX 33; EX 13).

Dr. Thomas M. Jarboe also reviewed the medical records on behalf of the employer and issued a report on December 21, 1995. Dr. Jarboe concluded that pneumoconiosis is present, but noted the difficulty in determining whether the claimant has a pulmonary or respiratory impairment because he consistently failed to give optimal effort on the pulmonary function studies. He found a mild obstructive defect attributable to smoking. He classified the claimant as totally and permanently disabled and stated that the claimant cannot do coal mine work. (DX 29). Dr. Jarboe is board-certified in internal and pulmonary medicine. (EX 13).

Dr. Younes was deposed on April 18, 1996. He explained that he observed clubbing of the fingernails[4] and enlarged lymph nodes when he examined the claimant. He attributed the enlarged lymph nodes to pneumoconiosis. He stated that there is no doubt that the x-ray showed changes consistent with pneumoconiosis, in light of the claimant's history. When questioned about the findings of several doctors that the claimant has sarcoidosis rather than pneumoconiosis, he stated that one can only diagnose sarcoidosis after performing a biopsy. A physician can observe a pattern that looks like sarcoidosis, but it must be confirmed with a biopsy. He testified that the claimant bled during the bronchoscopy, so that a biopsy was not done of the lymph nodes, but based on what was sampled, malignancy and tuberculosis were ruled out. Dr. Younes stated that coal workers' pneumoconiosis and sarcoidosis could look the same radiographically. He found that pneumoconiosis was more likely based on the claimant's history. He related the enlarged lymph nodes to coal workers' pneumoconiosis "most likely." Dr. Younes also noted that the claimant's chronic obstructive pulmonary disease, namely septal emphysema, could be caused by coal dust exposure or cigarette smoking. He disagrees with any diagnosis of

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[4] Dr. Dahhan also found clubbing in 1989. He testified that he did not note it in 1995 because, although there was still some clubbing present, it was not as prominent as it was in 1989. (DX 49, depo. at 25).

asthma. He stated that the claimant used to be a light smoker, but quit smoking fifteen years ago. When informed of the claimant's elevated carboxyhemoglobin levels, Dr. Younes attributed the elevation to factors other than smoking. He also explained that when attempting pulmonary function studies, the claimant tried hard, but was unable to sustain exhalation. He stated that even though he did not administer any valid pulmonary function study, he knew from his experience treating the claimant that the claimant was disabled. The arterial blood gas study he administered produced normal results. He found that the claimant was totally and permanently disabled regardless of whether the x-ray showed pneumoconiosis or sarcoidosis. He further stated that chronic bronchitis does not necessarily abate when the exposure to coal dust ceases, and that he has seen many cases in which it does not. Dr. Younes is board-certified in internal and pulmonary medicine. He sees the claimant approximately once a month. (DX 32, 41).

Dr. Kirk E. Hippensteel interviewed and examined the claimant on April 23, 1996 on behalf of the employer. He noted that there is evidence suggestive of pneumoconiosis radiographically, but it is basilar predominant which is atypical for coal workers' pneumoconiosis and pleural changes are also atypical for coal workers' pneumoconiosis. He concluded that he could not state with certainty what caused the x-ray abnormalities and therefore coal workers' pneumoconiosis could not be excluded. Dr. Hippensteel also noted a history consistent with chronic bronchitis, previous heart attacks, and back pain. He interpreted the pulmonary function study as showing a mild obstruction with very mild improvement after bronchodilation. The arterial blood gas test showed moderate hypoxemia at rest. Dr. Hippensteel determined that the claimant's mild airflow obstruction should not prevent him, from a pulmonary standpoint, from performing his job requirements as a section foreman. He did note that the claimant's medical problems as a whole may prevent his work in the mines. Dr. Hippensteel then reviewed other medical records. He stated that these records supported his examination opinion. Coal workers' pneumoconiosis is excluded as a cause of the pulmonary abnormality because the abnormality developed after the claimant left the mines and continued to smoke. (DX 34).

Dr. Hippensteel was deposed on May 10, 1996. He stated that when the claimant gave good effort, it could be seen that he had the functional capacity to continue his previous coal mine employment. He noted, however, that the claimant was totally disabled from a whole man perspective. Dr. Hippensteel did not find any respiratory disability due to coal dust exposure. (DX 39). Dr.

Hippensteel is board-certified in internal, pulmonary, and critical care medicine. (DX 39; EX 13).

Notes from the Green Valley Rural Clinic completed by Dr. Yasser Saloum, dated May 23, 1996, are in the record. Dr. Saloum made diagnoses of pneumoconiosis, unspecified; lung mass; coronary arteriosclerosis; history of myocardial infarction; diabetes mellitus type II, uncontrolled; history of allergy to penicillin; low vision, both eyes; degenerative joint disease. Acute bronchitis, heartburn, dyspnea, fever with chills, chronic cough, chronic bronchitis, and a rash were also diagnosed on occasion. Dr. Saloum considered the claimant to be a non-smoker. An x-ray was interpreted as showing increased interstitial markings B/L, probably chronic and a mild prominence of upper mediastinum. He noted that the claimant had chronic obstructive pulmonary disease, and that pulmonary function studies could not be performed due to the claimant's shortness of breath. He made no assessment of the claimant's pulmonary function. (DX 40, 41).

A CT scan was obtained on May 29, 1996. Dr. Paul Wheeler interpreted it as showing moderate paratracheal and minimal superior mediastinal and bilateral hilar adenopathy compatible with probable inflammatory disease. He also noted focal arteriosclerosis in both coronary arteries and moderate emphysema. He found no pneumoconiosis. (DX 45). Dr. Jerome Wiot's findings were similar. (DX 42). Dr. Fino agreed that there was no evidence of coal workers' pneumoconiosis, but noted diffuse bullous emphysema which compressed the lung tissue and caused increased markings. (DX 45).

Dr. D.H. Halbert read the May 29, 1996 CT scan as showing a ground glass opacity in the lungs, which is observed with several acute or active pulmonary processes, including alveolitis idiopathic pulmonary fibrosis, pneumonia, alveolar proteinosis, hypersensitivity pneumonitis, and sarcoidosis. (DX 40).

Another CT scan was obtained on June 4, 1996. Dr. Halbert noted small nodes in the mediastinum and right hilum. The nodes were normal in size, but the presence of them in the aortopulmonary window was abnormal. He also observed a diffuse ground glass opacity in both lungs. (DX 40, 41).

Dr. Wiot was deposed on October 23, 1996. He stated that he has vast experience in the field of pneumoconiosis and mentioned that he was on the Ohio Silicosis Commission in 1959 or 1960. Moreover, Dr. Wiot was on the team who developed the ILO Classification System

for x-rays, the classification system used to determined whether a person has pneumoconiosis. He was an original C reader and teaches the B reader course. Dr. Wiot had

the opportunity to review seven x-rays taken over a period of years and examined a CT scan. He stated that the claimant has a very abnormal chest with prominent adenopathy involving the hilar areas and right peritracheal area. The CT scan confirmed this observation and also showed a ground glass pattern to the lung and evidence of emphysema. He determined that the claimant does not have pneumoconiosis, explaining what should be apparent on an x-ray for a diagnosis of medical/classical pneumoconiosis. One factor that may be seen is:

> [S]o-called eggshell calcifications of the lymph nodes. Eggshell calcifications occur in other diseases than coal workers' pneumoconiosis, for instance, silicosis, which is the most common cause. But three percent of coal workers have them if they have advanced disease, and about 10 percent of silica workers, but you also see it in other diseases, such as sarcoid, treated Hodgkin's disease, amyloidosis. So it's not specific, but it is strong support if you have other reasons to suspect coal worker's pneumoconiosis ... .

Dr. Wiot diagnosed sarcoidosis, a granulomatous process of unknown origin. He stated that he has observed numerous patients with this condition because a colleague of his at the University of Cincinnati, where he teaches, specializes in sarcoid and therefore examines many patients with the condition. He stated that there was a possibility that the claimant has idiopathic pulmonary fibrosis, but that sarcoidosis was more likely. Dr. Wiot explained that adenopathy seen on x-rays is not related to pneumoconiosis, and that the CT scan revealed a ground glass pattern, which is also inconsistent with pneumoconiosis. He stated that both of those conditions are consistent with sarcoidosis. Dr. Wiot also testified that coal workers' pneumoconiosis may show some adenopathy pathologically. (DX 46).

Dr. Broudy issued a supplemental report on November 4, 1996 following his review of additional medical evidence. He stated that:

> Having reviewed this additional evidence, especially with regards to the CT scan reports and other x-ray interpretations, it is clear that this gentleman had some

> interstitial lung disease and probably chronic obstructive airways disease as well. The interstitial lung disease is not characteristic of coal workers' pneumoconiosis, at least by chest x-ray
>
> and CT scan findings. Even my own report, where I read the x-rays as Category 1/0 in 1984, shows that I found small irregular opacities of the s and t type which are not characteristic of coal workers' pneumoconiosis. I have not had an occasion to review any additional x-rays, to my knowledge. I believe the major difficulty here is that this gentleman does have some interstitial disease which is probably unrelated to pneumoconiosis. His lung impairment may be due to this interstitial disease unrelated to pneumoconiosis and airways obstruction due to cigarette smoking. It appears from the valid studies that he would retain the capacity to do his previous work or work requiring similar effort. I believe that he has chronic obstructive airways disease and interstitial pulmonary disease of undetermined etiology. Only a lung biopsy could definitely answer the question as to etiology of his interstitial lung disease. I am not sure that it is medically indicated and I suspect it would only be helpful from a legal standpoint. Therefore, I would not necessarily recommend that he have the lung biopsy because of its attendant risks.

(DX 48).[5]

Dr. Dahhan issued another report on November 6, 1996 after reviewing additional medical records. He concluded that the findings on the chest x-rays and CT scan are not consistent with pneumoconiosis. He deferred to those physicians who he described as experts in the field of pneumoconiosis, Drs. Wiot, Spitz, Wheeler, and Scott, who found no pneumoconiosis but attributed the x-ray markings to other diagnoses such as pulmonary sarcoidosis and idiopathic pulmonary fibrosis. Dr. Dahhan found an obstructive respiratory impairment as demonstrated by clinical examination findings. He determined that the claimant does not retain the physical capacity to do coal mine work due to asthmatic bronchitis associated with hyperactive airway disease. He stated that the

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[5] Compare Dr. Hippensteel's testimony in which he criticized Dr. Younes for not obtaining a biopsy (DX 51, depo. at 22-23).

pulmonary impairment and disability were not caused by coal dust exposure nor pneumoconiosis. (DX 48).

Dr. Fino issued a supplemental report on November 6, 1996 after considering additional medical information, including the CT scan. Dr. Fino concluded that the claimant's abnormality in lung function could not be accounted for by coal workers' pneumoconiosis. He revised his earlier opinion to state that the claimant did not have any coal dust related condition. He attributed the increased markings on the CT scan to diffuse bullous emphysema. He noted that the irregular markings in the lower and middle lung zones were inconsistent with CWP. He explained that the ILO classification system for x-rays categorizes all types of conditions that may be consistent with pneumoconiosis. He does not disagree with Dr. Wiot that either sarcoidosis or idiopathic pulmonary fibrosis were possible diagnoses. (DX 48).

Dr. James R. Castle reviewed medical records and issued a report on November 6, 1996 on behalf of the employer. Dr. Castle concluded that the claimant did not have coal workers' pneumoconiosis, had only a mild impairment related to tobacco smoke-induced chronic bronchitis, and was not totally disabled from a pulmonary or respiratory standpoint. He explained that "[s]ilicosis may be associated on some occasions with some degree of lymphadenopathy. However, coal workers' pneumoconiosis is not specifically associated with this." Dr. Castle then noted that the claimant may be totally disabled from coronary artery disease. He stated that even if pneumoconiosis were found, the claimant is not totally disabled due to pneumoconiosis, as the most recent valid pulmonary function study of May 1996 produced non-qualifying results. Dr. Castle is board-certified in internal and pulmonary medicine. (DX 48; EX 13).

Dr. Hippensteel issued a supplemental report on November 7, 1996 after considering additional medical evidence. He again concluded that the claimant had the capacity to perform his previous coal mine employment. He modified his previous opinion, based on the CT scan, to state that coal workers' pneumoconiosis is not the cause of the interstitial changes on the x-rays. He opined that sarcoidosis was the more likely cause. (DX 48).

Dr. Jarboe issued a supplemental report on November 8, 1996 following his review of additional medical evidence. He again determined that the claimant was totally disabled. He made this determination based on the medical records that show persistent wheezing and rhonchi despite aggressive therapy. He noted that the pulmonary function studies showed only a mild impairment, but the

records indicated that clinically the claimant is constantly symptomatic. He stated that with the requirements of the claimant's job, to crawl three hundred feet, lift two hundred pounds twenty times per day, and carry two hundred pounds for ten feet twenty times per day, could not be performed due to his wheezing and shortness of breath. He attributed the claimant's disabling

respiratory impairment to cigarette smoking and not to coal workers' pneumoconiosis or coal dust. After reviewing the CT scan and other medical data, Dr. Jarboe revised his previous opinion to state that the claimant did not have pneumoconiosis. Dr. Jarboe found it significant that both Drs. Halbert and Wiot noted a ground glass pattern, which cannot be caused by pneumoconiosis. He noted that sarcoidosis or idiopathic pulmonary fibrosis can cause such a pattern. Dr. Jarboe also stated that a CT scan is more accurate than an x-ray. Moreover, he pointed out, the claimant is responsive to Prednisone, a steroid, which is characteristic of a reversible airway disease associated with smoking and chronic obstructive pulmonary disease. Coal workers' pneumoconiosis is a fixed disease and not responsive to Prednisone. He also stated that:

> [C]oal workers' pneumoconiosis does not cause bullous emphysema except when there is marked dust retention in the lungs. The degree of emphysema present is proportionate to dust retention. The CT scan has confirmed only a ground glass appearance of the lung tissues and not nodular lesions which would indicate significant dust retention. This would indicate that the bullous emphysema which is present has been caused by causes other than dust inhalation. The most obvious cause is his heavy cigarette smoking.

(DX 48).

Dr. Dahhan was deposed on November 11, 1996. He discussed his examinations of April 7, 1989 and September 12, 1995 as well as the medical records that he reviewed. He noted that the claimant had an obstructive abnormality and did have a pulmonary impairment due to sarcoidosis. He attributed the claimant's obstructive abnormality to hyperactive airway disease or asthmatic bronchitis. He found no disability due to pneumoconiosis. Dr. Dahhan explained that the main source of carbon monoxide in humans is smoking. He noted that the average person should have less than 1 ½ to 2% hemoglobin combined with carbon monoxide. The claimant's was measured as 10.3% and 12%. (DX 49).

Dr. Castle was deposed on November 21, 1996 after reviewing the May 29, 1996 CT scan. He stated that the scan showed the absence of small, round regular nodules. Paraseptal emphysema was present, along with a widespread ground glass appearance in the middle lobes. Dr. Castle testified that the latter is typical of sarcoidosis. He believes the pleural changes most

likely represent fatty tissue, and testified that the pleural changes could be due to the steroid treatment. As to the lack of disability, Dr. Castle stated that:

> [A] single pO2 should not indicate permanent disability, particularly if it is not related to persistent decline and continuing fall, when we can see in this individual that there was variation. The one I indicated ... was 84.3, which is totally normal, and these kinds of variation are seen in patients that have a reduction of pO2 related to ventilation profusion mismatching, and that is associated with some mild degree of airways obstruction, and it may be related to his tobacco consumption or related to some inflammation due to his sarcoidosis.

(DX 52).

Dr. Hippensteel was also deposed on November 21, 1996 after reviewing the May 29, 1996 CT scan. He opined that the claimant has sarcoidosis, not coal workers' pneumoconiosis. He explained that:

> [Sarcoidosis] is an interstitial inflammatory disease that can also affect airways of the lung. It is a disease of the general public that we do not know the causation for it, but it is an immunologic process that in some ways has similarity to rheumatoid arthritis as an immunologic process ... .
>
> Rheumatoid arthritis is mainly a disease of the joints that causes inflammation there, and sarcoidosis has a predilection for lungs and lymph nodes. It turns out that sarcoidosis can affect joints and can affect other organs of the body, but the lungs and lymph nodes are the most common area to be involved, and that is why pulmonologists like me are the most common doctors to see and diagnose and treat sarcoidosis as a disease.

> It is a disease that starts, as we said, unexplicably and sometimes leaves unexplicably. It is responsive to steroids in regards to that medication helping decrease the inflammation that is there on a temporary basis, but ... steroids are not curative of the disease, so it still leaves some of it present, but it does help function during a time when a person has sarcoidosis bothering them.

He explained that sarcoidosis can cause bronchitis.

As to Dr. Younes' statement on the claimant's lack of good performance on pulmonary function studies being reflective of his disability, Dr. Hippensteel responded that:

> That has an anti-scientific ring to it, if you will. I have patients, for instance, that have chronic respiratory failure with lung diseases that can still give an adequate effort to show what function they have, and that effort, itself, even though it plays into what kind of peak function you can give when you have disease, has no relationship to the severity of the disease, unless you are almost, in fact, unable to breathe, which was certainly not the case in this man, and certainly, it's not a problem in people who are wanting to give good effort or who do not have other factors that make them not give good effort, but it is not a factor that should make it in any way think that this person is more disabled.

(DX 51).

Dr. Raghu R. Sundaram interviewed and examined the claimant on November 4, 1999. The smoking history was negative. Examination revealed rhonchi and wheezes. An x-ray was positive for pneumoconiosis, ½. A pulmonary function study was also obtained. Dr. Sundaram diagnosed coal workers' pneumoconiosis due to exposure to coal dust over twenty-six years, and related the results of the pulmonary function study to coal dust exposure. He found that the claimant was totally disabled from a pulmonary standpoint based on the shortness of breath with limited activity. (DX 62).

Dr. Dahhan interviewed and examined the claimant again on December 18, 1999. The smoking history was negative. Examination of the chest showed good air entry to both lungs with no crepitation or wheeze. An electrocardiogram revealed regular sinus rhythm with

normal tracings. The carboxyhemoglobin level was 5.3%, a value consistent with an individual smoking over a pack of cigarettes per day. An arterial blood gas test showed mild hypoxia with adequate ventilation at rest; the values were normal with exercise. A pulmonary function study revealed a mild obstructive ventilatory defect. The post-bronchodilator study was invalid due to poor effort, as was the diffusion capacity study. An x-ray was negative for pneumoconiosis, but positive

for cardiac enlargement with hyperinflated lungs. Dr. Dahhan additionally reviewed other medical records, including his previous report and deposition testimony. Dr. Dahhan concluded that:

> 1. There is insufficient objective data to justify the diagnosis of coal workers' pneumoconiosis based on the normal clinical examination of the chest, mild obstructive abnormality on spirometry testing, adequate blood gas exchange mechanisms at rest and after exercise and negative x-ray readings for pneumoconiosis.
>
> 2. Mr. Blankenship has a mild obstructive ventilatory defect as demonstrated by the various clinical and physiological parameters of his respiratory system.
>
> 3. From a respiratory standpoint, Mr. Blankenship retains the physiological capacity to continue his previous coal mining work as a foreman or job of comparable physical demand.
>
> 4. Mr. Blankenship's carboxyhemoglobin level was again elevated to a level consistent with an individual smoking over a pack per day, raising doubt regarding the accuracy of his history that he is a nonsmoker.
>
> 5. Mr. Blankenship's obstructive airway disease did not result from coal dust exposure or coal workers' pneumoconiosis. He has not had any exposure to coal dust since 1982, a duration of absence sufficient to cause cessation of any industrial bronchitis that he may have had. Furthermore, his family physician is treating him with multiple bronchodilators, indicating that he believes that his condition is responsive to such therapy. These findings are inconsistent with the permanent adverse affects of coal dust on the respiratory system.

> 6. Mr. Blankenship has coronary artery disease, low back pain and essential hypertension. All are conditions of the general public at large and are not caused by, contributed to or aggravated by coal dust exposure or coal workers' pneumoconiosis.

(DX 65).

Records from Green Valley Rural Clinics show that Dr. Saloum continued to treat the claimant through August 1997, when Dr. German De Joya took over his care. On April 4, 1997, the claimant's history of having "blood 'taken out' from him because he was making too much of it" was noted. Unspecified pneumoconiosis continued to be a diagnosis, but now "polcythemia secondary" was also a diagnosis. The claimant's most recent examination was on February 10, 2000. (CX 1).

Dr. De Joya interviewed and examined the claimant on December 22, 1999. The smoking history was negative. Examination revealed expiratory wheezes at both bases. An x-ray was positive for chronic interstitial fibrotic changes and small apical bullae. A pulmonary function study revealed a moderate to severe restrictive defect without bronchodilator response, and moderate to severe small airway disease. A pulse oximetry was also obtained. Dr. De Joya diagnosed coronary arteriosclerosis likely secondary to atherosclerosis; severe chronic obstructive pulmonary disease ("COPD") with a restrictive defect, secondary to pneumoconiosis; and chronic pleurisy secondary to pneumoconiosis. He stated that the claimant "will be unable to perform any coal mining work due to his respiratory impairment," with the impairment due primarily to COPD. On a separate sheet, he checked off that the claimant does have an occupational lung disease caused by his coal mine employment based on the pulmonary function study, chest x-ray, and CT scan. He also checked off that the claimant was totally disabled, with the cause being probably pneumoconiosis. (DX 71; CX 1, 2).

Dr. Sundaram interviewed and examined the claimant again on June 5, 2000. Examination revealed bilateral rhonchi, 1+ wheeze, and scattered crepitations. A pulse oximetry showed saturation of 85%. Dr. Sundaram diagnosed coal workers' pneumoconiosis, COPD, and diabetes mellitus. He stated that:

> Mr. Blankenship presented to the office complaining with increasing shortness of breath on very limited

> activity with a O2 saturation of 85%, therefore home oxygen therapy was prescribed to him. He is unable to indulge himself in any gainful employment. In addition to the medications he was on samples were given Unphyll 400 mg po daily, Pulmocort 1 to 2 puffs twice a day, and Verelan PM 200 mg PO daily.

(CX 1).

Dr. Hippensteel interviewed and examined the claimant again on July 10, 2000. The medical history included being on a nasal steroid inhaler for five to six years for chronic rhinitis with no history of allergies. The claimant explained to Dr. Hippensteel that the only reason he can think of as to why he has had an elevated carbon monoxide level in his blood is his gas water heater at home, which has been tagged by the gas company for not operating correctly. Examination revealed wheezes. An x-ray did not exclude simple pneumoconiosis with classification of 2/1, p/t, mostly in the mid lung zones, and bilateral pleural thickening from an unclear cause. A pulmonary function study was invalid due to suboptimal effort. Lung volumes showed mild restriction with some improvement post bronchodilator. The diffusion was severely reduced, but when corrected for the lung volume the claimant inhaled for the test, was only moderately decreased with some question of validity of values, referable to effort. An electrocardiogram did not show any significant abnormalities. An arterial blood gas test revealed mild hypoxemia at rest. The carboxyhemoglobin level was 5.7%, consistent with a person who smokes a pack of cigarettes per day and was much more significantly elevated than expected from exposure to second-hand smoke. Dr. Hippensteel concluded that:

> This man has evidence of interstitial lung infiltrates radiographically. The pulmonary technician and myself had trouble getting adequate valid efforts for determination of function under spirometry, but it could be stated that he has no more than mild ventilatory impairment from any cause, even with this difficulty getting efforts. His diffusion is reduced, partially affected by his elevated carboxyhemoglobin level, which raises questions about his veracity regarding his smoking behavior. It would be unexpected that his malfunctioning gas heater at home would raise his level enough from its malfunction to be this level this long after leaving his house. This man has severe hypertension and coronary artery disease which

> are causes for shortness of breath. He also has musculoskeletal pain for which he is on medication without good relief. These nonpulmonary problems impair him as a whole man, and, in fact, were the reason he was not exercised on this examination, but they have nothing to do with his prior coal dust exposure. From a pulmonary standpoint alone, he does not have sufficient impairment to keep him from working at heavy manual labor. His arterial blood gases on this examination show that he does not have
>
> sufficient hypoxemia to need oxygen, and does not have evidence that he has developed cor pulmonale.

After reviewing additional medical data, Dr. Hippensteel added that:

> It appears that this man ... has shortness of breath just as much from his heart disease and hypertension, as from any lung problems. Although I cannot exclude pneumoconiosis completely as a diagnosis in this man, I think it can be stated with a reasonable degree of medical certainty that he does not have coal workers' pneumoconiosis as a diagnosis. It appears that any mild pulmonary impairment that he had is referable to bronchitis, which is likely to be associated with his continued carboxyhemoglobin elevations, which are most likely related to continued smoke exposure from tobacco products, and possibly added to by bronchial inflammation from sarcoidosis, which also participates in his chest x-ray abnormalities. The bilateral pleural plaques remain specifically unexplained, but could relate to old chest trauma with evidence of prior right rib fracture. This man gave no history of asbestos exposure that could refer to these pleural changes.

(EX 2).

Dr. Broudy reviewed records again and issued a report on August 31, 2000. He diagnosed "some type of interstitial pulmonary disease which [is] nonoccupational in nature." He found that the claimant had the respiratory capacity to resume his former coal mine employment. Dr. Broudy related the mild obstruction to chronic obstructive airways disease from cigar-ette smoking and some predisposition to asthma or bronchospasm. (EX 4).

Dr. Dahhan also performed another record review and issued a report on September 5, 2000. He now concluded that:

> 1. There is equivocal radiological data for the diagnosis of simple coal workers' pneumoconiosis. However, the CT of the chest was read by Drs. Spitz, Wheeler, Wiot and Scott, experts in the radiological field of occupational lung diseases, as not showing evidence of pneumoconiosis. This leads me to conclude
>
> that he has no convincing radiological data to support the diagnosis of coal workers' pneumoconiosis.
>
> 2. Mr. Blankenship has an obstructive ventilatory impairment, which waxes and wanes as demonstrated by various values on different occasions.
>
> 3. Mr. Blankenship has an obstructive ventilatory defect that is responsive to bronchodilator therapy as indicated by his treating physician treating him with multiple oral and inhaled bronchodilator medications.
>
> 4. Mr. Blankenship does not have the respiratory physiological capacity to perform heavy manual labor based on the most recent pulmonary function studies and arterial blood gases.
>
> 5. Mr. Blankenship's carboxyhemoglobin level has been elevated on more than one occasion, raising doubt regarding the validity of his statement that he is a nonsmoker.
>
> 6. Mr. Blankenship's obstructive ventilatory defect did not result from coal dust exposure or coal workers' pneumoconiosis. Instead, it waxes and wanes in wide swings, as demonstrated by the various values on his spirometry tests, indicating that it is not fixed in nature. Furthermore, his response to various broncho-dilator therapies including Theophylline, Beta II agonists and inhaled steroids, as indicated by the treatment regimen noted by his treating physician, also indicates that it is not fixed in nature. All of these parameters lead me to conclude that his condition is not due to a coal dust induced lung disease.

(EX 6).

Dr. Jarboe issued a supplemental report on October 10, 2000 after reviewing additional medical data. He found further support for his opinion that the claimant does not have coal workers' pneumoconiosis. He believed that the claimant has airflow obstruction but that it was difficult to assess the degree because of repeatedly suboptimal performance on the pulmonary function studies. He opined that the apparent restriction was due to a markedly elevated residual volume (186%) and air trapping. Dr. Jarboe then stated that:

> Despite the fact that the functional studies are only mildly to moderately impaired, as previously stated the records indicate that he is constantly symptomatic with wheezing and shortness of breath. The additional medical records, especially those from Green Valley Rural Clinic, would support this position. They repeatedly described wheezing and rhonchi in the chest. As stated, he was said to have poor functional status and to be short of breath ambulating only a short distance. It should be kept in mind that these findings were present even in the face of aggressive medical management with inhaled bronchodilators, Prednisone and inhaled corticosteroids.
>
> It is my reasoned opinion that despite functional studies that exceed the Federal limits for disability in coal miners, I do not feel that Mr. Blankenship could do his last coal mining job. He described that as requiring him to occasionally crawl 300 feet, lift 200 pounds 20 X day and carry 200 pounds for ten feet 20 X a day. In my opinion, the previous records and the most recently available records would indicate that he cannot do this.
>
> I feel that this disabling impairment is the result of cigarette smoking and not coal dust inhalation. Despite Mr. Blankenship's denial of smoking, it would be extremely unusual to repeatedly record elevated levels of carboxy hemoglobin in a nonsmoker. This has been done over a span of several years in different laboratories.
>
> There also appears to be an asthmatic component to Mr. Blankenship's pulmonary problem. He has been treated with oral and inhaled steroids and intensive bronchodilator therapy. These are the types of treatment used in asthma. Coal worker's pneumoconiosis tends to cause a fixed impairment and one would not expect these

> medications to be used in this disease entity nor one would expect a response.

(EX 10).

Dr. Fino issued a supplemental report on October 11, 2000 after reviewing additional medical data. His opinions remained the same. (EX 9).

Dr. Castle's supplemental report was issued on October 12, 2000 after he reviewed additional medical data. His opinions also remained the same. (EX 8).

Dr. Hippensteel issued yet another report on October 18, 2000 after reviewing additional medical data. His opinions remained the same. (EX 11).

Dr. Ben V. Branscomb reviewed approximately seven hundred pages of medical documents and issued a report on October 13, 2000. He noted that "[i]n 1983 he was hospitalized for, he said, shortness of breath. He was found to have too much red blood and four units removed. (This means he had been diagnosed as hav- ing polycytemia vera.)" Later in his report, he stated that "[t]hroughout most of the years Mr. Blankenship had been diagnosed as p. vera. Some, such as Dr. Myers, pointed out that the etiology of the excess blood cells had really not been established. In any case, Mr. Blankenship had received the treatment for p. vera." As to the x-ray readings, Dr. Branscomb commented that:

> [T]he ground glass increase resembling a fine and uniform interstitial process is also very typical in patients with polycythemia vera. This is because of the massive increase in total blood volume with distension of all of the small pulmonary vessels by excess blood. I did not see any indication by the readers who most suspected sarcoid that they were aware that polycythemia vera had been diagnosed.
>
> The reports, discussions, and depositions by Doctors Wheeler, Scott, and Wiot discuss in a scholarly way the differential diagnosis and why these distinguished radiologists concluded the changes were not consistent with CWP.

> Based on comparison of all the interpretations, the findings on re-reading of films, and the qualifications of the readers, the x-rays do not support a diagnosis of CWP.

Dr. Branscomb noted that the pulmonary function tests were sufficient to indicate that there is no restrictive impairment and no worse than mild obstructive impairment. As to the arterial blood gas tests, he summarized that:

> [M]any of the oxygen values have been normal and some have shown a mild reduction at rest. At no time are the values low enough to meet the Federal disability standards. I note that the oxygen tension seems to have become higher after Mr. Blankenship lost a great deal of weight. The oxygen values are entirely consistent with the combination of his obesity and his polycythemia vera. The latter is one of the conditions which most notably produces mild and varying levels of hypoxemia.

He concluded that the claimant was not totally disabled from a pulmonary standpoint. (EX 12).

Dr. Dahhan was deposed again on November 6, 2000. He reiterated his opinions, except that now he stated that the claimant was not totally disabled based on the objective studies. (EX 15).

Dr. Hippensteel was deposed again on November 10, 2000. He reiterated his opinions, and provided a copy of the chapter "Sarcoidosis" from Interstitial Lung Disease (3rd Ed.), B.C. Decker, Inc., London, 1998. (EX 16).

The medical evidence submitted with the claimant's first claim for benefits is set forth in Judge Roketenetz's July 23, 1990 Decision and Order - Denial of Benefits (DX 23.53), Judge O'Neill's Decision and Order - Denial of Benefits (DX 56), and the Joint Stipulation of Medical Evidence (JX 1).

Modification

Section 725.310 provides that a claimant may file a petition for modification within one year of the last denial of benefits. Modification petitions may be based upon a change in condition or a mistake in a determination of fact. 20 C.F.R. § 725.310(a).

In deciding whether the claimant has established a change in conditions, I must "perform an independent assessment of the newly submitted evidence, in conjunction with evidence previously submitted, to determine if the weight of the new evidence is sufficient to establish the element or elements which defeated entitlement . . .." Napier v. Director, OWCP, 17 BLR 1-111, 1-113 (1993). *See also* Nataloni v. Director, OWCP, 17 BLR 1-82, 1-84 (1993).

In deciding whether the prior decision contains a mistake in a determination of fact, I must review all the evidence of record, including evidence submitted since the most recent denial. New evidence, however, is not a prerequisite to modification based upon a mistake of fact. Nataloni, 17 BLR at 1-84; Kovac v. BCNR Mining Corp., 14 BLR 1-156, 1-158 (1990), *aff'd on recon.* 16 BLR 1-71, 1-73 (1992). *See also* O'Keefe v. Aerojet-General Shipyards, 404 U.S. 254, 257 (1971). Rather, the factfinder is vested "with broad discretion to correct mistakes of fact, whether demonstrated by wholly new evidence, cumulative evidence, or merely further reflection on the evidence initially submitted." O'Keefe, 404 U.S. at 257.

The claimant has requested modification of Judge O'Neill's denial of benefits to show a mistake of fact in the finding that the claimant's total disability was not due to pneumoconioisis. The employer has requested modification of that same decision to show a mistake of fact in the finding that a material change in conditions was established under § 725.309(d), arguing that the claimant is not totally disabled. I address first the § 725.309 (d) issue of a material change in conditions.

Duplicate Claim

In cases where a claimant files more than one claim and a prior claim has been finally denied, later claims must be denied on the grounds of the prior denial unless the evidence demonstrates "a material change in condition." 20 C.F.R. § 725.309 (d). The United States circuit courts of appeals have developed divergent standards to determine whether "a material change in conditions" has occurred. Because the claimant last worked as a coal miner in the state of Kentucky, the law as interpreted by the United States Court of Appeals for the Sixth Circuit applies to this claim. *Shupe v. Director, OWCP*, 12 BLR 1-200, 1-202 (1989).

Under the Sixth Circuit's standard for determining the existence of a material change in conditions, an administrative law judge must consider all of the new evidence, both favorable and unfavorable, to determine whether the miner has proven at least one of the elements of entitlement that previously was adjudicated against him. If a claimant establishes the existence of one of these elements, he will have demonstrated a material change in condition as a matter of law. Then, the administrative law judge must consider whether all the evidence of record, including evidence submitted with the prior claim, supports a finding of entitlement to benefits. *Sharondale Corp. v. Ross*, 42 F.3d 993, 997-98 (6th Cir. 1994). *See Lisa Lee Mines v. Director, OWCP*, 86 F.3d 1358, 1363 (4th Cir. 1996).

Because the claimant filed his application for benefits after March 31, 1980, this claim shall be adjudicated under the regulations at 20 C.F.R. Part 718. Under this part of the regulations, claimant must establish by a preponderance of the evidence that he has pneumoconiosis, that his pneumoconiosis arose from coal mine employment, that he is totally disabled, and that his total disability is due to pneumoconiosis. Failure to establish any of these elements precludes entitlement to benefits. *See Anderson v. Valley Camp of Utah, Inc.*, 12 BLR 1-111, 1-112 (1989).

As previously mentioned, the claimant's first claim for benefits was denied for failure to establish total disability and total disability due to pneumoconiosis. With the instant claim, Judge O'Neill found that the claimant had established total disability, and thus a material change in conditions. I agree with that determination.

Although the arterial blood gas tests did not continue to reveal qualifying values and the pulmonary function studies have shown a mild impairment, the treatment progress notes and medical opinions show that the claimant's clinical status is critical in determining his ability to work. The claimant is constantly symptomatic despite numerous medications. In this regard, I give great weight to the claimant's treating physician, Dr. Younes, who offered a documented and well-reasoned opinion on the issue. I also give great weight to the opinion of Dr. Jarboe, who went beyond the test values and looked at what was realistic for this claimant. Drs. Fritzhand, Fino, Sundaram, and DeJoya also found the claimant to be totally disabled. I find that these opinions outweigh the opinions of Drs. Broudy, Dahhan, Hippensteel, Castle and Branscomb, who focused on the variability of the test values and the non-qualifying status of most of them. Those studies do not measure the claimant's

coughing, his chest pain from coughing, the amount of wheezing, nor the impact these symptoms have on his ability to function as a worker.

Dr. Dahhan initially found the claimant to be totally disabled (considering both the study results and the clinical symptoms), then changed his mind (with his opinion showing no thought about the clinical symptoms at that time), then found him to be totally disabled again (based on the objective studies), and then found him to not be totally disabled (again based on the objective studies).

I therefore find no mistake in the previous determination that the claimant is totally disabled from a pulmonary standpoint, and has therefore established a material change in conditions. As such, the claimant is entitled to a de novo review of his claim for benefits.

Pneumoconiosis and Causation

Under the Act, pneumoconiosis is defined as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b). Section 718.202(a) provides four methods for determining the existence of pneumoconiosis: X-ray evidence, biopsy or autopsy evidence, application of a presumption, and medical opinion evidence. §§ 718.202(a)(1)-(4).

Under the provisions of § 718.202(a)(1), chest x-rays that have been taken and evaluated in accordance with the requirements of § 718.102 may form the basis for a finding of the existence of pneumoconiosis if classified in Category 1, 2, 3, A, B, or C under an internationally-adopted classification system. An x-ray classified as Category 0, including subcategories 0/-, 0/0 and 0/1, does not constitute evidence of pneumoconiosis. Under §718.202(a)(1), when two or more x-ray reports are in conflict, consideration must be given to the radiological qualifications of the physicians interpreting the x-rays.

The x-ray readings and testimonies of the physicians indicate that the claimant does have x-ray changes which can be classified as consistent with pneumoconiosis, Category 1 to 2, with one reader (Dr. Rubenstein) rating the changes in 1995 as Category 3.

Some readers declined to classify the changes as pneumoconiosis because they concluded, based on the shape and location of the opacities, that the etiology of the changes was not coal workers'

pneumoconiosis. They related the changes to sarcoidosis, idiopathic pulmonary fibrosis, or another possible cause. However, § 718.202(a)(1) does not concern itself with the shape of the opacities or their location in the parenchyma; rather, it looks at the size of the opacities. The opacities in this case clearly meet the size requirements for Category 1 or greater.

In any event, it is noted that the various readings revealed a mixture of shapes - p, q, s, and t - not just irregular ones.[6] Many readers found rounded opacities. The readers also differed as to location, with many readers finding opacities scattered throughout the lungs. Further, the classified x-ray readings that were rendered do not show any significant variation per reader. The readings were as follows:

Physician	First Reading	Subsequent Readings
Anderson	Category 1	
Cole	½	½
Myers	2/1	2/2
Penman	2/2	
Quillin	1/1	
Clarke	2/2	
Mettu	2/2	
Sargent	1/0	½
Poulos	2/2	2/2
Rubenstein	3/2	
Mathur	2/3	
Dahhan	½	2/2
Shipley	2/1	
Hippensteel	2/2	2/2, ½, 2/1, 2/1
Castle	½	2/1
Sundaram	½	
Broudy	2/3	

Thus, apart from the temporary decrease from Category 2 to Category 1 observed by Dr. Hippensteel, the readers have found that the opacities remained stable or slightly increased.

As to the apparent pleural changes seen on x-ray, Drs. Castle and Hippensteel have concluded that they represent fatty tissue, not

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[6] Printed on the back of the x-ray forms is a statement that p, q, and r are rounded opacities; s, t, and u are irregular.

pleural plaques. Drs. Scott and Castle also indicated that the pleural changes may be due to the steroid treatment. In this regard, it is immaterial whether coal workers' pneumoconiosis causes pleural changes. Not every change in the lung has to be due to coal dust exposure in order for the presence of pneumoconiosis to be recognized.

For these reasons, I find that the x-ray evidence is positive for changes consistent with pneumoconiosis under § 718.202 (a)(1). The additional arguments on etiology will be discussed under §718.203(b).

A bronchoscopy was undertaken in 1994 by Dr. Younes. However, it did not provide any information on the existence of pneumoconiosis. Subsequently, Dr. De Joya tried unsuccessfully to convince the claimant to have a biopsy since he has a swelling mass or lump in his chest. (CX 1). The record is therefore devoid of any biopsy evidence to consider.

Under § 718.202(a)(3), a claimant may prove the existence of pneumoconiosis if one of the presumptions at §§ 718.304 to 718.306 applies. Section 718.304 requires x-ray, biopsy, or equivalent evidence of complicated pneumoconiosis. Because the record contains no such evidence, this presumption is unavailable. The presumptions at §§ 718.305 and 718.306 are inapplicable because they only apply to claims that were filed before January 1, 1982, and June 30, 1982, respectively. Because none of the above presumptions applies to this claim, the claimant has not shown pneumoconiosis pursuant to § 718.202(a)(3).

Section 718.202(a)(4) provides that a claimant may establish the existence of pneumoconiosis is a physician exercising reasoned medical judgment, notwithstanding a negative x-ray, finds that he suffers from pneumoconiosis.

Apart from x-ray changes, the claimant also has been diagnosed with chronic obstructive pulmonary disease (chronic bronchitis/emphysema). If significantly related to coal dust, this condition would meet the statutory definition of pneumoconiosis at § 718.201. Dr. Mettu did not address an etiology. Dr. Fritzhand was under the impression that the claimant was a non-smoker. I find these two opinions entitled to no weight on the issue.

Drs. Younes, Sundaram, and De Joya named coal dust as the cause. Drs. Jarboe, Hippensteel, Broudy, Dahhan, Fino, and Castle related the condition to smoking. Drs. Dahhan and Jarboe also opined that there was a component of asthma, which Dr. Younes disagreed with.

Dr. Hippensteel also testified that sarcoidosis can cause bronchitis. The issue of sarcoidosis will be addressed under §718.203(b).

As to smoking as a cause, the claimant testified that he never smoked more than a few cigarettes a day, and that his smoking was confined to when he was a teenager. While that testimony is somewhat contradictory to his previous testimony that the smoking occurred over a number of years, it is also consistent, particularly as to the amount that was smoked each day. His statements to physicians and his testimonies reveal a smoking history of a few cigarettes per day, beginning as a teenager and ceasing sometime in his adulthood, possibly as late as 1984. Some physicians recorded a history of one-quarter or one-half pack of cigarettes per day. Accepting the claimant's statements regarding his smoking history as true, there still is the question of an elevated carboxyhemoglobin level on at least two occasions since 1984. The claimant named two possible causes: a faulty water heater and secondhand smoke.

Dr. Younes dismissed the elevated carboxyhemoglobin levels, stating that it was not an indication that the claimant was still smoking and noting that there could be many causes.

Nowhere in the record is it indicated that the claimant smelled of smoke, or had discolored teeth or finger tips from smoking. See, e.g., September 14, 1995 report of Dr. Dahhan, wherein he opined that the claimant was smoking two packs or more of cigarettes per day, yet no other cigarette-related finding is noted. (DX 22B). Nowhere in the record is there an indication that any treating physician thought the claimant was smoking and/or advised him to quit.

As to coal dust exposure as a cause, the parties stipulated that the claimant was a coal miner for at least ten years. All of his work was underground at the face. The claimant worked alongside the other miners as a section foreman. He testified that the working conditions were very dusty.

Dr. Dahhan stated that the "waxing and waning" seen on the pulmonary function studies was an indication that the condition causing the problem was not related to coal dust exposure. However,

I find that this part of his opinion is not well-reasoned, as it fails to pinpoint what the "waxing and waning" amounts to once variable effort on the studies, as well as the differing effects of the various medications, are taken into account.

Dr. Dahhan also stated that the chronic bronchitis was not due to coal dust exposure because any industrial bronchitis would have resolved shortly after exposure to coal dust ceased. Dr. Younes strongly disagreed with this based on his experience.

Weighing this evidence, I find that the claimant has established that his chronic obstructive pulmonary disease (chronic bronchitis/emphysema) is due to coal dust exposure. The physicians who related this condition to cigarette smoking speculated that the claimant had a significant smoking history and was still smoking. While smoking may be the major cause of high levels of carbon dioxide in humans, the evidence is undisputed that a high carboxyhemoglobin level can be caused by factors other than smoking. A connection between the claimant's high carboxyhemoglobin level and smoking is not substantiated. No physician found that the claimant's exposure to secondhand smoke was causative. Therefore, I find the opinions connecting the COPD (chronic bronchitis/emphysema) and smoking to be undocumented.

Great weight is placed on the opinions of Drs. Younes and De Joya who, respectively, was and is the claimant's treating physician. They concluded that coal dust exposure is a cause of the claimant's COPD (chronic bronchitis/emphysema). They observed the claimant on many occasions, and rendered documented and well-reasoned opinions.

I therefore find that the medical opinions show that the claimant has statutory pneumoconiosis, namely chronic obstructive pulmonary disease, consisting of chronic bronchitis and emphysema, due to coal dust exposure. § 718.202(a)(4).

Weighing all of the evidence, both like and unlike, on the issue of pneumoconiosis, I find that the claimant has established that he has pneumoconiosis, both medical and statutory.

Pursuant to § 718.203(b), the claimant is entitled to invocation of the rebuttable presumption that his pneumoconiosis arose from his coal mine employment. The evidence in rebuttal on the cause of the claimant's COPD/chronic bronchitis was discussed under § 718.202(a)(4). I herein discuss the evidence and argument related to the cause of the x-ray changes found to be consistent with pneumoconiosis under § 718.202(a)(1).

Dr. Branscomb opined that the x-ray changes are due to polycythemia vera, which the claimant suffered with in the 1980s and for which he underwent treatment. However, I find this opinion outweighed, as Dr. Branscomb was the only physician to consider any connection between the polycythemia vera and x-ray changes. Other internists and pulmonologists, namely Drs. Broudy, Fino, Myers, Guberman, and Dahhan (DX 23), as well as Dr. Saloum, were aware of the polcythemia vera diagnosis and treatment, and yet raised no concern of any connection in their reports in the prior claim nor in the current claim. I therefore find that the evidence on polcythemia vera does not rebut the § 718.203(b) presumption that the claimant's pneumoconiosis arose from his coal mine employment.

The majority of physicians are of the opinion that the changes represent sarcoidosis or, less likely, idiopathic pulmonary fibrosis. Drs. Jarboe, Hippensteel, and Fino initially diagnosed coal workers' pneumoconiosis, but changed their minds upon reviewing the CT scan reports. Drs. O'Neill, Wright, Dahhan, Broudy, Castle, Wheeler, Scott, Shipley, and Wiot have maintained that the x-ray changes are not due to coal workers' pneumoconiosis.

On the other hand, Drs. Myers, Penman, Clarke, Anderson, Saloum, Younes, Mettu, Fritzhand, Sundaram, and De Joya related the x-ray changes to pneumoconiosis. Dr. Saloum was the only one not to specify a connection between the x-ray changes and coal dust; his records indicate "unspecified" pneumoconiosis.

Significantly, three of the latter physicians, Drs. Saloum, Younes, and De Joya, have been the claimant's treating physicians. These three physicians practice together at Green Valley Rural Clinics. The CT scan reports of Dr. Halbert, obtained by them, were a part of the medical data they considered in rendering their opinions.

Dr. Baxter stated that the opacities were "suggestive of this representing chronic changes from exposure to inorganic dust." (DX 23.163). Dr. Guberman did not make an x-ray finding. (DX 23.382).

I find that the evidence on sarcoidosis, idiopathic pulmonary fibrosis, or another cause, to be insufficient to rebut the §718.203(b) presumption. The evidence shows that there are numerous conditions which can appear similarly to coal workers' pneumoconiosis on an x-ray and, as Dr. Younes testified, there is no absolute certainty absent a biopsy. Thus, this record contains several possibilities - coal workers' pneumoconiosis, sarcoidosis, idiopathic pulmonary fibrosis, polcythemia vera, and asbestosis. A

presumption avoids having to go back and forth on all the possibilities, and not being able to resolve all doubts on causation in favor of the claimant.

But even without the presumption, the evidence shows that the claimant's x-ray changes are due to coal workers' pneumoconiosis. His treating physicians found so, and their opinions are documented and well-reasoned. They are the ones who prescribed the steroids and have followed their effects. The physicians diagnosing sarcoidosis placed great weight on the finding of adenopathy; yet Dr. Scott has raised the possibility of the adenopathy and the pleural fat being due to the steroid treatment. Dr. Younes also testified that adenopathy can be seen with coal workers' pneumoconiosis, which Dr. Wiot disputed that to the extent that it would not be associated on x-rays, but could be seen pathologically. The physicians diagnosing sarcoidosis also placed great weight on the ground glass appearance in the lungs. They stated that the CT scans show no round nodules in the lungs, and that the ground glass opacities were mistaken for nodules on the x-rays. However, Drs. Younes and De Joya diagnosed coal workers' pneumoconiosis and COPD related to coal dust exposure despite the finding of a ground glass appearance.

I therefore find that the evidence establishes that the claimant's pneumoconiosis arose from his coal mine employment.

Causation of Total Disability

Finally, the claimant must also establish that his total disability is due to pneumoconiosis. 20 C.F.R. § 718.204(b). To satisfy this requirement, the United States Court of Appeals for the Sixth Circuit requires a claimant to prove that his totally disabling respiratory impairment is due "at least in part" to his pneumoconiosis. *Adams v. Director, OWCP*, 886 F.2d 818, 825 (6th Cir. 1989).

The claimant is totally disabled based on his clinical symptoms and the objective test results, which are due to his two pulmonary conditions: the changes on the x-rays, found to be medical/classical pneumoconiosis, and the COPD (chronic bronchitis/emphysema), found to be statutory pneumoconiosis. Drs. Younes, Sundaram, and De Joya connected all these factors in their reports. I therefore find that the claimant has established that his total disability is due to pneumoconiosis.

Date of Entitlement

In the case of a miner who is totally disabled due to pneumoconiosis, benefits commence with the month of onset of total disability. Where the evidence does not establish the month of onset, benefits begin with the month that the claim was filed. 20 C.F.R. § 725.503(b). The instant claim was filed on January 26, 1995. Dr. Younes first expressed an opinion of total disability in May 14, 1996, however, he diagnosed severe coal workers' pneumoconiosis before then. Dr. Fritzhand found the claimant to be totally disabled on September 8, 1995. These opinions do not provide an exact date of onset, and do not show that the claimant was not totally disabled when he filed his claim. Consequently, the claimant shall receive benefits commencing January 1, 1995, based on the month the claim was filed.

Attorney's Fee

Claimant's counsel has thirty days to submit an - application for an attorney's fee. The application shall be prepared in strict accordance with 20 C.F.R. §§ 725.365 and 725.366. The application must be served on all parties, including the claimant, and proof of service must be filed with the application. The parties are allowed thirty days following service of the application to file objections to the fee application.

In reaching the above conclusion, I have applied the various versions to the regulations recently promulgated by the Department of Labor. 65 Fed. Reg. 79920-80107 (Dec. 20, 2000). I note this decision was rendered only after giving due consideration to the arguments of the parties, both the new and old regulations, and existing core law, inconcluding the new regulations at issue in *National Mining Association v. Chao*, No: 100CN0386(EGS) (D.C. Dist.)

ORDER

Based on the above findings, Kentucky Carbon Corporation is hereby ORDERED to pay the following:

1. To the claimant, Jack R. Blankenship, all benefits to which he is entitled under the Act augmented by reason of one dependent, commencing January 1, 1995;

2. To the claimant, all medical and hospitalization benefits to which he is entitled, commencing January 1, 1995.

A
JOSEPH E. KANE
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 CFR § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this Decision and Order by filing a Notice of Appeal with the Benefits Review Board at Post Office Box 37601, Washington, D.C. 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.